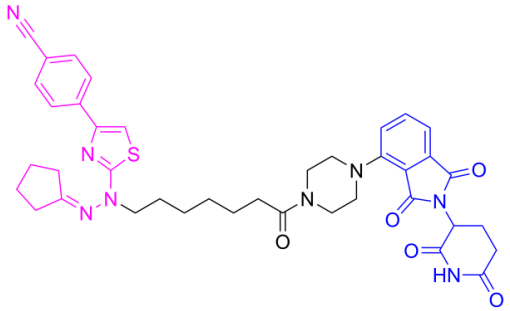
Product Data Sheet

Product Name: NP1192
Cat. No.: GC80928

Chemical Properties

Cas. No.	2966791-41-1		
Formula	$C_{39}H_{42}N_8O_5S$	M.Wt	734.87
Solubility	DMSO: 100 mg/mL (136.08 mM; Need ultrasonic)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Background

NP1192 is a potent, selective PROTAC NAT10 degrader. NP1192 promotes NAT10 ubiquitination and degradation. NP1192 inhibits ac4C modification. NP1192 demonstrates dual inhibition of hypoxia-driven glycolysis and immunosuppression via NAT10/ HIF-1α/PD-L1 axis disruption,

achieving superior antitumor efficacy and synergizing with anti-PD-L1 both in vitro and in vivo. NP1192 can be used for ovarian, cervical, and glioblastoma cancer research [1] [2] [3]. (Pink: NAT10 ligand; Blue: Cereblon ligand; Black: linker).

In Vivo, NP1192 (25 mg/kg, i.p., every 2 days for a week) synergizes with anti-PD-L1 to inhibit tumor growth, suppress glycolysis, and enhance CD8+ Teff cell immunity in U14-luc xenograft mouse model[1].

In Vitro, NP1192 (0-20 μM, 24-48 h) induces NAT10 degradation in SiHa (human) and U14 (murine) CCa cell lines in a dose-and time-dependent manner, through the ubiquitin-proteasome system (UPS) rather than via lysosomal pathways[1]. NP1192 (36 h) inhibits cervical cancer (SiHa) cell growth with an IC_{50} of 7.814 μM (76.2 μM in resistant cells) and additionally shows dose-dependent efficacy in organoids, with IC_{50} values of 9.947, 3.048, and 13.76 μM for ovarian (OV), cervical (CESC), and glioblastoma (GBM) models, respectively[1]. NP1192 (20 μM, 0-72 h and 2 weeks) significantly reduces cell viability, induces cell cycle arrest, and suppresses invasion and clonogenic growth in CCa cells[1]. NP1192 (0.195-50 μM, 1-8 days) induces a pronounced reduction in organoid size and number across tumor models (ovarian, cervical, and glioblastoma), while the corresponding normal organoids remained unaltered, indicating minimal cytotoxicity to nonmalignant tissues[1]. NP1192 (20 μM, 36 h) reprograms the hypoxic tumor microenvironment (TME) and metabolism in CCa cells by degrading NAT10 and thereby impairing HIF-1α expression, which disrupts glycolysis as evidenced by reduced glucose uptake and lactate production alongside elevated ATP levels[1].

References:
[1]. Ao, K., et al. Targeted NAT10 Degradation by PROTAC NP1192 Suppresses Hypoxia-Adaptive Glycolysis and Reinvigorates CD8+ Effector T-Cell Function for Synergistic Cancer Immunotherapy. ACS Central Science. 2025.
[2]. Zhou Q, et al. PROTAC technology for sensitizing melanoma to immune checkpoint therapy[J]. Journal of Experimental & Clinical Cancer Research, 2026.
[3]. Xiong J, et al. Functional roles and mechanisms of NAT10-mediated RNA ac4C modification in normal development and cancer progression. Front Cell Dev Biol. 2026 Mar 6;14:1795359.